UNITED STATES DISTRICT COURT

EASTERN DISTRICT OF WASHINGTON

NEIL HENRICKSEN AND MAURITA HENRICKSEN, Plaintiffs, vs. CONOCOPHILLIPS COMPANY, Defendant.	NO. CV-07-224-JLQ **MEMORANDUM OPINION AND ORDER ON MOTIONS; ORDER GRANTING DEFENDANT'S MOTION FOR SUMMARY JUDGMENT**

The court has had under advisement the following eleven motions.

1. Defendant's Motion to Exclude Opinions of Dr. Caton Based on Disclosure Violations (Ct. Rec. 67);

2. Defendant's Motion in Limine to Limit the Testimony of Plaintiffs' Treating Physicians (Ct. Rec. 71);

3. Defendant's Motion to Exclude the Testimony of Plaintiffs' Expert Witness Frank Gardner (Ct. Rec. 75);

4. Defendant's Motion to Exclude Dose Opinions of Expert Sawyer due to Disclosure Violations (Ct. Rec. 79);

5. Defendant's Motion to Exclude Testimony of Plaintiffs' Expert Witness William Sawyer (Ct. Rec. 83);

6. Defendant's Motion to Exclude the Testimony of Plaintiffs' Expert Witness Marco Kaltofen (Ct. Rec. 87);

7. Defendant's Motion to Exclude Causation Opinions Based on Unreliable Epidemiological Evidence (Ct. Rec. 91)

8. Defendant's Motion for Summary Judgment on General Causation (Ct. Rec. 95)

9. Defendant's Motion for Summary Judgment on Specific Causation (Ct. Rec. 100);

10. Defendant's Motion to Exclude Plaintiffs' Expert John Caton, M.D. (Ct. Rec. 127); and

11. Plaintiff's expedited Motion to Supplement the Record (Ct. Rec. 160).

I. INTRODUCTION

Neil Henricksen ("Henricksen"), a former gasoline tanker truck driver, and his wife brought suit against the Defendant (among others), ConocoPhillips (Defendant or "Conoco"), alleging that Henricksen's acute myelogenous leukemia (AML) was caused by his occupational exposure to benzene and benzene-containing products, including Defendant's gasoline. Plaintiffs assert products liability claims for negligence, strict liability and breach of warranty.

By motions to exclude/motions *in limine* Defendant seeks to have the court decide whether Plaintiffs can reliably establish a medical or scientific link between exposure to gasoline and AML and whether Plaintiffs' admissible evidence can establish that Henricksen's exposure to Defendant's gasoline proximately caused his illness. Focusing both on Plaintiffs' general causation and specific causation case, Defendant seeks to exclude the testimony and reports of Plaintiffs causation experts Marco Kaltofen, Ph.D., Frank Gardner, M.D., William Sawyer, Ph.D., and Peter Infante, Ph.D., as well as Henricksen's treating physicians. In contesting the reliability of the Plaintiffs' proposed evidence Defendants have submitted reports and testimony of their own expert witnesses, David Pyatt, Ph.D., Ethan Natelson, M.D, John Spencer (an industrial hygienist), and David H. Garabrant, M.D., MPH.

Specifically, Defendant argues that Plaintiffs cannot establish that exposure to benzene as a component of gasoline increases the risk of developing AML. Defendant also asserts that Plaintiffs can not demonstrate, through reliable testimony and evidence that Henricksen's occupational exposure to benzene in gasoline was sufficient to cause his

AML. Defendant challenges the methodology employed by Plaintiffs' experts and argues their opinions have no scientific basis, are not supported by the material facts of this case, are not supported by reliable studies, have not been tested, have not been subjected to peer review, and are inadmissible under the Federal Rules of Evidence and as explained by the United States Supreme Court in *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993) and subsequent decisions of the federal courts. In addition to its *Daubert* motions, Defendant has moved to strike any testimony on the subject of causation by treating physician John Caton and the dose opinions of William Sawyer based upon Plaintiffs' alleged failure to timely disclose these individuals as experts.

Defendant's motions for summary judgment argue that because the opinions of Plaintiffs' experts are unreliable and therefore inadmissible, Plaintiffs can not demonstrate that gasoline caused Henricksen's AML. In their opposition to the summary judgment motions, Plaintiffs attempt to demonstrate a genuine issue regarding causation by proffering the testimony of the experts and the scientific data that Defendant has challenged in its motions *in limine*. Therefore, whether Plaintiffs can defeat the summary judgment motions turns on the extent to which Defendant's motions *in limine* are meritorious.

The parties have set forth an extensive evidentiary record, which includes the reports of each expert, excerpts from depositions, and numerous publications. Defendant requested an evidentiary hearing and oral argument. The court granted this request. The parties, by stipulation (Ct. Rec. 231), agreed not to present any live witness testimony at the hearing and agreed to rely solely on the written record. Appearing at the hearing on behalf of the Plaintiffs was Jackson Schmidt. Appearing on behalf of the remaining Defendant were Christopher Weiss, Brett Young, Stephen Dillard, and Leslie Weatherhead.

After careful consideration of the extensive briefing on the motions, the responses, the replies, the evidence submitted, the applicable law, and counsel's argument, the court finds the disputed testimony fails scrutiny under *Daubert.* Thus for the reasons that

follow, Defendants' Motions For Summary Judgment are **GRANTED**.

II. THE RECORD EVIDENCE

Before turning to the legal analysis, the court summarizes the facts before it and the evidence presented with respect to these issues. In doing so the court will review the Plaintiffs' experts proffers and the substance of their opinions.

Plaintiff Neil Henricksen worked as a gasoline truck driver from 1973 -2003. As part of his duties, Henricksen loaded petroleum fuels, including gasoline, refined and sold by, among others, the Defendant ConocoPhillips. He loaded fuels at terminals in and around Spokane, Washington, including at Defendant's Spokane terminal. Apparently, during his tenure as a truck driver, Henricksen drove a top-loading truck that was filled through a hatch at the top of the tank. It is alleged he regularly spilled gasoline on his skin. Half of his time was spent loading diesel fuel as opposed to gasoline. Plaintiffs do not contend exposure to diesel fuel was a contributing factor here. Conoco's Spokane terminal allegedly did not have a vapor recovery system to prevent exposure to gasoline fumes. Ct. Rec. 147, Ex. 6. It is alleged the terminal also had a roof and partial walls on two of the four sides, such that fumes did not as easily dissipate as would in "open" terminals. In August 2003, at age 61, Henricksen was diagnosed with a form of cancer, Acute Myelogenous Leukemia (AML). He received chemotherapy and his AML went into remission for two years. In 2005, his AML returned and he was again placed into a second treatment with chemotherapy. He received a bone marrow transplant from a sibling and at the time of the filing of the present motions, he was again in remission.

1. AML

AML is one of the most common types of leukemia in adults in the U.S., with approximately 13,000 new cases diagnosed each year. Ct. Rec. 104, Ex. A [*Gardner*] at 51:4-8. AML afflicts people of all ages and from all cultures, though the risk of AML increases with age. Unlike asbestosis caused by asbestos exposure, AML is not a disease that implicates its cause. While there are various known causes of the disease, the majority of cases have no known cause. One way AML has been classified is according

to presumed causation. Cases can either be termed idiopathic or *de novo* (primary, endogenous), meaning onset without external or environmental stimulus, or *secondary* (event-related, exogenous) events which could be related to exposure to chemotherapy or radiation interventions in addressing preexisting hematologic disorders, or exposure to environmental toxins, including benzene. Ct. Rec. 104, Ex. B [*Natelson Aff.*]. The majority (80-90%) of all adult AML cases are *de novo* or idiopathic, with no readily identifiable cause. Ct. Rec. 104, Ex. E [*Pyatt Aff]* at 20; *Natelson*, ¶ 17 . Studies of patients with AML have shown the underlying biology of these two categories of AML is usually different. Either cytogenetic or a distinct pattern of chromosomal aberrations have been considered characteristic findings in nearly ninety percent of all secondary AML, which includes AML caused by exposure to benzene as opposed to gasoline containing benzene. *Id.* In *de novo* AML cytogenetic abnormalities are observed only in approximately fifty percent of the time. *Pyatt Aff.*, ¶ 12. There was no evidence of chromosomal abnormality in Mr. Henricksen's case.

In addition, secondary forms of AML are commonly preceded by myelodysplastic syndrome (MDS) and are more resistant to standard treatment than de novo AML. *Id.* Mr. Henricksen's AML was not preceded by MDS and after standard chemotherapy, remained in remission for two years.

In addition, there are different forms or variants of AML which are assigned designations (M0-M7) based on "morphological and cytogenetic similarities" within each subtype. *Pyatt Aff.*, ¶ *12.* The M1 variant of AML is a frequently observed subtype comprising of 20% of all *de novo* AML cases. Mr. Henricksen's subtype was M1.

In the 10-15 years immediately prior to his diagnosis in 2003, Henricksen was predominately transporting diesel fuel. Plaintiffs have focused on the period from 1976-1983/1984 when Henricksen worked at the Parkwater Terminal as the years he experienced the majority of exposure to gasoline. Defendants' experts claim the scientific studies have shown that exposures occurring within 10-15 years of clinical onset contribute to risk of AML, but those in more distant past do not. *Pyatt Aff.*, ¶ 13; Ct. Rec.

104, Ex. V [*Garabrant Aff.*] at ¶ 8. Plaintiffs experts disagree.

Given the presentation and clinical and laboratory features of Henricksen's disease, according to Defendant's experts, there is no scientific way to separate the AML affecting Henricksen from the *de novo* AML occurring in people with no particular exposure to chemicals. *Pyatt Aff., ¶ 15; Natelson, ¶ 17.* Taken collectively, on medical probability alone, Defendant's experts conclude Henricksen's AML is far more consistent and must be considered *de novo* rather than secondary due to occupational exposure to the benzene in gasoline. *Id.*

2. Gasoline

Gasoline is a complex mixture of chemical substances (as many as 150 distinct compounds and other additives). It is undisputed that one component of gasoline is benzene. Conoco's gasoline contained between 1 and 4.9% benzene. Ct. Rec. 147, Ex. 7 [Conoco MSDS sheets] at 1. The chemical composition of gasoline vapor differs from the chemical composition of gasoline liquid. Benzene vapor makes up a small fraction (0.5-0.7%) of total gasoline vapor.

Benzene vapor concentration is usually measured as parts of benzene per million parts of air (ppm). Ct. Rec. 94, Ex. V [Garabrant Aff.] at ¶ 7. Thus if there was one molecule of benzene in one million molecules of air, the benzene concentration would be one part per million. *Id.* In order to measure a person's cumulative exposure to benzene, the average concentration during a typical workday is multiplied by the duration in years and is expressed in parts per million-years, or ppm-years. *Id.* This is sometimes referred to as a "time-weighted average" or TWA.

The parties agree that scientific studies provide clear evidence of a causal relationship between occupational exposure to benzene and benzene-containing solvents and the occurrence of AML. Benzene exists in the environment everywhere and humans are exposed to benzene on a daily basis. Ct. Rec. 151, Ex. B (Sawyer Rebuttal Report) at 23. Benzene exists in air, water, soil, and in our food. Low exposures to which every human being is subjected, is often, and alternatively, referred to as “background

exposure" or "ambient exposure". No one, including the Plaintiffs' experts, proffers an opinion that this level of exposure creates an increased risk of the development of AML. Everyone, including the Plaintiffs' experts, agrees that something greater is required. The argument in this *Daubert* challenge, in part, revolves around the question of how much greater quantity of exposure is necessary to permit the causal attribution of AML to a particular benzene exposure.

Though there is no unanimous agreement as to the threshold, both the Defendants and Plaintiffs agree the medical-scientific literature supports the conclusion that cumulative benzene exposure at levels between 50 and 500 ppm-years, can result in AML. It is Plaintiffs' expert's contention however, that far lower levels (as low as 1.5 ppm/years) of long term exposure to benzene are linked with a significantly elevated risk of AML.

Just as the toxicity of benzene has been evaluated and studied, so has the toxicity of gasoline. Both parties experts recognize that no authoritative source (organization or regulatory agency) has identified gasoline as cancer-causing. It has been named as a "probable carcinogen" because animal studies with fuel vapors have demonstrated a significant increase in kidney cancer among male rats and liver cancer in female mice. The U.S. Occupational Safety and Health Administration (OSHA) does not mandate any occupational exposure limit for workers exposed to gasoline, whereas OSHA has promulgated a 1 ppm workplace exposure limit for benzene. The American Conference of Governmental Industrial Hygienists (ACGIH) has a recommended, allowable, 8-hour work day exposure limit for gasoline of 300 ppm, whereas the current recommendation for exposure to benzene is .5 ppm. General Causation SJ, Ex. B., *Rose* at 108:13-15. The National Institute for Occupational Safety and Health's (NIOSH) recommended exposure limit is 0.1 ppm for an 8-hour TWA and 1 ppm for short-term exposure (NIOSH 1992b).

The Defendant's experts contend the observed toxicity of gasoline is different from the observed toxicity following exposure to pure benzene. While bone marrow toxicity is

almost universally found following high-dose benzene exposure in experimental animals, Defendant's experts say no studies of any species, including humans, have reported bone marrow toxicity following even chronic exposure to high levels of unleaded gasoline. Pyatt, ¶ 11. Some toxicological experts theorize that so called "competitive inhibition" between benzene and the other compounds found in gasoline mitigate the potential carcinogenic properties of the small amounts of benzene present in gasoline. *Pyatt, ¶ 12; Natelson*, ¶ 18. Toxicologically, Defendant's contend it is unsupportable that exposure to benzene as **a component of gasoline** plays a role in the development of AML. *Pyatt*, ¶ 11. Defendant's experts contend there is no scientific or medical literature to support the proposition that there is a sufficient level of benzene, as a constituent ingredient of gasoline, to result in a risk of AML by virtue of exposure to gasoline.

Plaintiffs' expert epidemiologist, Peter Infante, on the other hand, states in his report that the "toxicity of gasoline to the bone marrow has mirrored the toxicity of benzene even though the literature related to gasoline has lagged that of benzene." Ct. Rec. 149.

3. Plaintiffs' Experts

Henricksen was treated by physicians John Caton and Jeanie Nichols of Cancer Care Northwest and George Earle Georges and Andrew Kominsky, of Seattle Cancer Care Alliance. Dr. Caton was Mr. Henricksen's primary treating physician who apparently told the Henricksens and also presently believes that his AML was likely caused by occupational exposure to gasoline.

Plaintiffs argue in opposition to the Defendant's motions that they have presented sufficient reliable and valid evidence of both general and specific causation to raise a triable issue. Plaintiffs evidence on causation includes the testimony of expert witnesses Peter Infante, an epidemiologist, Frank Gardner, a medical doctor, William Sawyer, a toxicologist, and Marco Kaltofen, a civil engineer. Plaintiffs contend that their expert evidence demonstrates that even at low levels, exposure to benzene in gasoline increases the risk of developing AML. Below is a brief description of each expert and his

conclusion.

William Sawyer, Ph.D., is a toxicologist board certified in forensic medicine, toxicology and pharmacology. He has authored on benzene poisoning. Sawyer calculates Henricksen's annual benzene exposure in this case as 1.118 ppm and his cumulative dose as 8.9 ppm-years. In his opinion, he concludes that "to within a reasonable degree of toxicological certainty...Mr. Henricksen was exposed to dangerously high levels of carcinogenic benzene..." and that "to within a reasonable degree of toxicological certainty...Mr. Henricksen's chronic toxic exposure excursions...to benzene while top loading primarily contributed to the development of his AML."

Marco Kaltofen is a registered professional engineer and environmental scientist who has performed environmental, workplace and product investigation. He was hired by Plaintiffs to calculate Henricksen's estimated exposure to benzene. Kaltofen estimate's Henricksen's "unadjusted daily benzene exposure" as 0.38 ppm. He then adjusts this number based upon features of Henricksen's work and the length of time he performed the work, resulting in a cumulative dose estimate of 1.6625 ppm-years. He then multiplies that number by 5 to accommodate the fact that Henricksen worked at a terminal with a roof to come up with a dose estimate of 8.3125 ppm-years.

Frank Gardner, M.D. is a Clinical Professor of Medicine in the Division of Hematology-Oncology in Galveston, Texas. He has studied and published on the treatment of leukemia. Gardner opines that on a more probable basis than not, benzene exposure was "a major cause of Mr. Henricksen's acute myelogenous leukemia." Ct. Rec. 148, Ex. A. "Mr. Henricksen, from his prolonged exposure to benzene in gasoline, initiated mutagenic changes in the bone marrow leading to his leukemia." He bases this conclusion solely upon Mr. Henricksen's description of his occupation and his belief that chronic low exposure to benzene can cause AML.

Peter Infante, Ph.D., is an epidemiologist and is the managing member and President of Peter F. Infante Consulting, L.L.C., an environmental and occupational health consulting firm. In 1977, he conducted the first study of benzene exposed workers

in the production of Pliofilm at Goodyear plants. In this case, based upon his review of the scientific literature, he opines that 1) both occupational exposure to benzene and to gasoline, which contains an average 1-2% benzene, are associated with an elevated risk of developing AML and 2) "Mr. Henricksen's 29 years of occupational exposure to gasoline was a substantial contributing factor and the most likely cause of his development of AML." Ct. Rec. 149.

III. LEGAL STANDARDS

A. DAUBERT

In the seminal case of *Daubert v. Merrell Dow Pharmaceuticals, Inc*. 509 U.S. 579, 113 S.Ct. 2786, 125 L.Ed.2d 469 (1993), the Supreme Court cast trial judges in the role of "gatekeeper[s] in determining whether to admit or exclude expert evidence[ ]" in accordance with Rule 702. *See Dukes v. Wal-Mart, Inc.*, 509 F.3d 1168, 1179 (9th Cir. 2007) (internal quotation marks omitted). The Daubert Court "held that Federal Rule of Evidence 702 commands the primary focus for courts evaluating the admissibility of expert testimony." *Cooper v. Brown*, 510 F.3d 870, 942 (9th Cir. 2007). That Rule provides in part that "[i]f scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact issue," an expert "may testify thereto." Fed.R.Evid. 702. Before a witness may come "before the jury cloaked with the mantle of an expert[ ]" under Rule 702, the Ninth Circuit has cautioned that "care must be taken to assure that a proffered witness truly qualifies as an expert, and that such testimony meets the requirements of [that] Rule[.]" *Jinro America Inc. v. Secure Investments, Inc*., 266 F.3d 993, 1004 (9th Cir. 2001). Thus, as a threshold matter, in accordance with Rule 702 the court must determine whether the proffered witness is "qualified as an expert by knowledge, skill, experience, training, or education[.]" Fed.R.Evid. 702; *see also Daubert v. Merrell Dow Pharm., Inc*., 43 F.3d 1311, 1315 (9th Cir. 1995) (" Daubert II")(whether proffered expert testimony is admissible "only arises if it is first established that the individual[ ] whose testimony is being proffered [is][an] expert[ ] in a particular ... field[ ]"). Defendant does not challenge the expert

qualifications of any of Plaintiffs' experts, except the treating physicians.

Once a court makes the "preliminary" determination under Fed.R.Evid. 104(a) that a witness qualifies as an expert, the focus shifts to that witness's proffered testimony. Rule of Evidence 702 permits a witness to give expert testimony if "(1) the testimony is based upon sufficient facts or data, (2) the testimony is the product of reliable principles and methods, and (3) the witness has applied the principles and methods reliably to the facts of the case." Fed.R.Evid. 702 . As interpreted by the *Daubert* Court, there is a two-part inquiry under Rule 702 for determining the admissibility of proffered expert opinion testimony. Expert testimony must be "not only relevant, but reliable." *Daubert*, 509 U.S. at 589, 113 S.Ct. 2786. This requires consideration of whether:

(1) the reasoning or methodology underlying the testimony is scientifically valid (the reliability prong); and

(2) whether that reasoning or methodology properly can be applied to the facts in issue (the relevancy prong).

Daubert v. Merrell Dow Pharms., Inc., 509 U.S. 579, 592-93, 113 S.Ct. 2786, 125 L.Ed.2d 469 (1993).

Reliability Prong. In determining reliability, a court may consider a number of factors including: (1) whether the theory can be and has been tested; (2) whether it has been subjected to peer review; (3) the known or potential rate of error; and (4) whether the theory or methodology employed is generally accepted in the relevant scientific community. *Id.* at 593-94. However, "Daubert' s list of specific factors neither necessarily nor exclusively applies to all experts or in every case." *Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 141, 119 S.Ct. 1167, 143 L.Ed.2d 238 (1999). "[F]ar from requiring trial judges to mechanically apply the Daubert factors ... judges are entitled to broad discretion when discharging their gatekeeping function." *United States v. Hankey*, 203 F.3d 1160, 1168 (9th Cir. 2000) (citing *Kumho*, 526 U.S. at 150-52).

The court need not admit an expert opinion that is connected to the underlying data "only by the ipse dixit of the expert." *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997).

It may exclude such testimony if it determines "that there is simply too great an analytical gap between the data and the opinion proffered." *Id*. "The trial court's gate-keeping function requires more than simply taking the expert's word for it." *Daubert v. Merrell Dow Pharms., Inc.*, 43 F.3d 1311, 1319 (9th Cir. 1995) (" Daubert II "). In addition, "any step that renders [the expert's] analysis unreliable ... renders the expert's testimony inadmissible. This is true whether the step completely changes a reliable methodology or merely misapplies that methodology." *In re Silicone Gel Breast Implants Products Liability Litigation*, 318 F.Supp.2d 879, 890 (DC Cal. 2004). Something doesn't become scientific knowledge just because it's uttered by a scientist; nor can an expert's self-serving assertion that his conclusions were derived by the scientific method be deemed conclusive. *Daubert* II, at 1315-16. "[T]he expert's bald assurance of validity is not enough. Rather, the party presenting the expert must show that the expert's findings are based on sound science, and this will require some objective, independent validation of the expert's methodology." *Id*., at 1316.

Relevance Prong. The relevance prong under *Daubert* means that the evidence will assist the trier of fact to understand or determine a fact in issue. *Daubert*, 509 U.S. at 591-92. "The gatekeeping inquiry must be 'tied to the facts' of a particular 'case.' " *Kumho*, 526 U.S. at 150 (*quoting Daubert*, 509 U.S. at 591)). "Encompassed in the determination of whether expert testimony is relevant is whether it is helpful to the jury, which is the 'central concern' of Rule 702." *Mukhtar*, 299 F.3d at 1063 n. 7 (citation omitted). Federal Rule of Evidence 702 states that "[i]f scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue," an expert "may testify thereto," Fed.R.Evid. 702.

Burden of Proof. "It is the proponent of the expert who has the burden of proving admissibility." *Lust v. Merrell Dow Pharmaceuticals, Inc.*, 89 F.3d 594, 598 (9th Cir. 1996). Admissibility of the expert's proposed testimony must be established by a preponderance of the evidence. *See Daubert*, 509 U.S. at 592 n. 10 (*citing Bourjaily v. United States*, 483 U.S. 171, 175-76, 107 S.Ct. 2775, 97 L.Ed.2d 144 (1987)). The party

presenting the expert must demonstrate that the expert's findings are based on sound principles and that they are capable of independent validation. *Daubert II*, 43 F.3d at 1316.

Defendant's *Daubert* motions do not challenge the credentials of plaintiffs' experts (other than the treating physicians). All of the experts utilized in this case have impeccable curriculum vitaes, and many have extensive experience testifying as experts. Accordingly, the only *Daubert* issue to decide whether Plaintiffs' experts have employed a reliable methodology in reaching the conclusions they propose to offer to the jury at trial.

B. SUMMARY JUDGMENT

Summary judgment is not "a disfavored procedural shortcut but rather [it is] an integral part of the Federal Rules as a whole, which are designed 'to secure the just, speedy, and inexpensive determination of every action.' " *Celotex Corp. v. Catrett*, 477 U.S. 317, 327, 106 S.Ct. 2548, 91 L.Ed.2d 265 (1986) (quoting Fed.R.Civ.P. 1). It is appropriate "if the pleadings, depositions, answers to interrogatories, and admissions on file, together with the affidavits, if any, show that there is no genuine issue as to any material fact and that the moving party is entitled to a judgment as a matter of law." Fed.R.Civ.P. 56(c). In evaluating a motion for summary judgment, the court views the evidence and draws all reasonable inferences therefrom in the light most favorable to the nonmoving party. *United States v. Diebold, Inc.*, 369 U.S. 654, 655, 82 S.Ct. 993, 8 L.Ed.2d 176 (1962).

The movant's burden is "discharged by showing ... that there is an absence of evidence to support the nonmoving party's case." *Celotex*, 477 U.S. at 325, 106 S.Ct. 2548. Only after the movant meets its initial burden does any obligation on the part of the nonmovant arise. *Id.* at 323, 106 S.Ct. 2548. Nevertheless, once the movant has met this initial burden, the opposing party must present evidence establishing a material issue of fact. *Id*. at 325, 106 S.Ct. 2548. The nonmoving party must go "beyond the pleadings" and present evidence designating "specific facts showing that there is a genuine issue for

trial." *Id.* at 324, 106 S.Ct. 2548.

An issue of fact is "material" if it is essential to the proper disposition of the claim. *Anderson v. Liberty Lobby, Inc*., 477 U.S. 242, 248, 106 S.Ct. 2505, 91 L.Ed.2d 202 (1986). In essence, the inquiry is "whether the evidence presents a sufficient disagreement to require submission to a jury or whether it is so one-sided that one party must prevail as a matter of law." *Id*. Further, a jury question does not exist because of the presence of a mere scintilla of evidence; rather, there must be significant probative evidence tending to support the complaint and conflict in substantial evidence to create a jury question. *Fazio v. City & County of San Francisco*, 125 F.3d 1328, 1331 (9th Cir. 1997).

IV. ANALYSIS

A. CAUSATION

Because the Defendant has not challenged the qualifications of Plaintiffs' experts, the court focuses on the substance of Plaintiffs' proffered expert testimony. In particular, whether the methodology by which the experts have reached their conclusions is reliable and whether those conclusions will assist the trier of fact in resolving an issue of fact in this case. In resolving this issue "we must look to the governing substantive standard..." *Daubert v. Merrell Dow Pharms.*, 43 F.3d 1311, 1320 (9th Cir. 1995).

Courts in toxic tort cases often separate the causation inquiry into general causation and specific causation. To survive summary judgment on their allegations, Plaintiffs must show (on a more probable than not basis) that Henricksen was exposed to a product that is capable of causing a particular condition he complains of in the general population (general causation), and that his exposure did in fact result in those injuries (specific causation). *Jaros v. E.I. DuPont* (*In re Hanford Nuclear Reservation Litig*.), 292 F.3d 1124, 1133 (9th Cir. 2002).

In *Hanford,* the Ninth Circuit explained that the general causation inquiry was "whether exposure to a substance for which a defendant is responsible, such as radiation at the level of exposure alleged by plaintiffs, is capable of causing a particular injury or

condition in the general population." *Id*. Plaintiffs argue this case is not one where general causation is an issue because there is no dispute that benzene is capable of causing the illness Henricksen suffers from, and gasoline contains benzene. Since these facts are undisputed, Plaintiffs argue this case becomes a question of only of dose. According to Defendant, however, gasoline and benzene are not the same, and as a matter of science, the chemical composition and toxicity is different from pure benzene. These competing characterizations of the issue raise the first question the court will decide in its analysis: what is the product at issue here - benzene or gasoline that contains benzene? Is there a difference and, if so, is the difference meaningful in the *Daubert* context?

Defendant calls this a gasoline case. Plaintiffs call this a benzene case and say any other characterization is a "red herring", a "cheap parlor trick" and a "misdirection" of the Defendant. The framing of the issue defines the Plaintiffs' burden, so it is important. This is a product's liability action and Defendant's product is gasoline. It is undisputed that Henricksen's exposure was to the mixture gasoline, not simply to the substance benzene. Benzene and gasoline, as evidenced by the unrebutted testimony of Defendant's expert, are not one in the same product. Ct. Rec. 104, Ex. E [Pyatt Aff.] at 14-18. Gasoline is a mixture of chemicals, which contains as a small component, benzene. There are scientific studies devoted to the study of benzene (and benzene exposure from gasoline vapors) and a whole body of scientific studies devoted to the study of gasoline. In fact the common approach to the evaluation of the gasoline exposures is to measure the individual toxic compounds, including benzene. Because gasoline exposure is a source of benzene exposure, evaluations of both gasoline and its toxic component benzene are obviously relevant to the Plaintiffs' case.

At the same time, the court can not simply presume that the qualitative toxic and carcinogenic effects of benzene from *any source* are the same. If it is possible to extrapolate from studies of benzene or other benzene-containing products conclusions regarding gasoline, then it will be incumbent upon Plaintiffs to explain and demonstrate why the extrapolation is scientifically proper. This is even more true given the vast

difference in threshold limit values for these substances; studies in the record such as *Glass*[1], indicating that "exposure to benzene concentrate resulted in a higher risk of leukemia than exposure to the same amount of benzene encountered in a more dilute form such as in gasoline"; and Defendant's unrebutted expert evidence that the toxicity of gasoline has been tested and is "categorically different than the toxicity observed following exposure to pure benzene." *Pyatt*, at ¶ 11; Ct. Rec. 104, Ex M[Jamall, I. and Willhite, C., *Is Benzene Exposure from Gasoline Carcinogenic*, 10 J. ENVIRON. MONIT.176 (2008)]. The general causation question before the court is whether exposure to the benzene-component of gasoline is capable of causing AML.

Specific causation is defined simply as "whether exposure to an agent was responsible for a given individual's disease." Federal Judicial Center, Reference Manual on Scientific Evidence 396 (2d ed. 2000). In determining whether an alleged chemical exposure caused a particular disease or illness, an expert must establish the following criteria: (1) the toxic substance at issue must have been demonstrated to cause in humans the disease or illness suffered by the plaintiff; (2) the individual must have been exposed to a sufficient amount of the substance in question to elicit the health effect in question; (3) the chronological relationship between exposure and effect must be biologically plausible; and (4) the likelihood that the chemical caused the disease or illness in an individual should be considered in the context of other known causes. See David L. Eaton, Scientific Judgment and Toxic Torts-A Primer in Toxicology for Judges and Lawyers, 12 J.L. & Pol'y 5, 38-40 (2003) ("Eaton")**.**

Several appellate courts have held that an expert who seeks to opine on specific causation must pay careful attention to the dose-response relationship (that is, the relationship in which a change in amount, intensity, or duration of exposure to a chemical is associated with a change in risk of disease) and the amount of exposure the plaintiff allegedly suffered. *See, e.g., McClain v. Metabolife Int'l, Inc.*, 401 F.3d 1233, 1241-42

[1] Glass, Deborah, *Leukemia Risk Associated with Low-Level Benzene Exposure*, 14:5 EPIDEMIOLOGY 569 (2003).

(11th Cir. 2005); *Mitchell v. Gencorp, Inc.*, 165 F.3d 778, 781 (10th Cir.1999) ("It is well-established that a plaintiff in a toxic tort case must prove that he or she was exposed to and injured by a harmful substance manufactured by the defendant.... In order to carry this burden, a plaintiff must demonstrate 'the levels of exposure that are hazardous to human beings generally as well as the plaintiff's actual level of exposure to the defendant's toxic substance before he or she may recover.' ") (citation omitted); *Wintz by and through Wintz v. Northrop Corp*., 110 F.3d 508, 513 (7th Cir. 1997); *Wright v. Willamette Indus., Inc.*, 91 F.3d 1105, 1106 (8th Cir. 1996); *Allen v. Pa. Eng'g Corp*., 102 F.3d 194, 199 (5th Cir.1996) ("Scientific knowledge of the harmful level of exposure to a chemical, plus knowledge that the plaintiff was exposed to such quantities, are minimal facts necessary to sustain the plaintiff's burden in a toxic tort case.").

However, as Plaintiffs point out, it is not always necessary for a plaintiff to quantify exposure levels precisely or use the dose-response relationship, provided that whatever methods an expert uses to establish causation are generally accepted in the scientific community. While precise or exact information concerning dosage or the dose-response relationship is not always required, the boundaries of allowable expert testimony are not so wide as to permit an expert to testify as to specific causation without having any measurements of a plaintiffs' exposure to the allegedly harmful substance. *Hardyman v. Norfolk & Western Ry. Co.*, 243 F.3d 255, 264 (6th Cir. 2001).

Plaintiffs' experts have drawn conclusions about both the capacity of benzene (and benzene in gasoline) and its particular effect in Henricksen's case. Defendant's have attempted to draw sharp lines between the general and specific causation inquiries, filing separate summary judgment motions on each. The court notes, however, that while the division between general and specific causation is frequently helpful, the core issue that the jury will have to address in the case is whether Henricksen's exposure to gasoline was a substantial cause of his AML. In the end, the *Daubert* question comes down to whether Plaintiffs' experts' conclusions can validly assist the jury in deciding this question. Certainly, Defendant's concession that its product contains a known carcinogen --

benzene -- does not excuse Plaintiffs from having to show the benzene contained in Defendant's gasoline is capable of causing the illness at issue.

The court must begin by examining each of the experts' conclusions to determine if the method the expert has used to reach that conclusion is reliable. Then the court must examine those conclusions that are sufficiently reliable to be admissible and determine if, taken collectively, they form a sufficient causal chain to aid the trier of fact in reaching the ultimate conclusion on causation: whether Henricksen's exposure to gasoline was a substantial factor in his development of AML.

c. Categories of Evidence

To prove their case on general and specific causation, the categories of evidence relied upon by the parties' experts includes animal studies, differential diagnosis, and epidemiological studies.

Differential diagnosis is the process of elimination that physicians routinely use to identify the "most likely" cause of a particular individual's illness. It is an acceptable source of data on specific causation. *Hall v. Baxter Healthcare Corp.*, 947 F.Supp. 1387, 1413 (D.Or 1996). By examining the patient's symptoms, medical history, diagnostic test results, etc., a doctor can eliminate alternative causes and reach a conclusion about the most likely cause of a particular patient's condition. It is important to note, however, that differential diagnosis cannot demonstrate general causation, because it assumes, without proving, that all of the potential causes considered are capable of causing the condition at issue. “Indeed differential diagnosis assumes that general causation has been proven for the list of possible causes it eliminates[.]” *Id.*

As for epidemiological studies, “The field of epidemiology addresses the incidence, distribution and etiology (causation) of disease in human populations by comparing individuals exposed to a particular agent to unexposed individuals to determine whether exposure increases the risk of disease.” *In re Silicone Gel Breast Implants Products Liab. Lit.*, 318 F.Supp.2d 879, 892 (C.D.Cal. 2004). Scientists use “relative risk” to identify an association between, for example, the ingestion of a drug and

a disease.

> For example, if a study found that 10 out of 1000 women with breast implants were diagnosed with breast cancer and 5 out of 1000 women without implants (the "control" group) were diagnosed with breast cancer, the relative risk of implants is 2.0, or twice as great as the risk of breast cancer without implants. This is so, because the proportion of women in the implant group with breast cancer is 0.1 (10/1000) and the proportion of women in the non-implant group with breast cancer is 0.05 (5/1000). And 0.1 divided by 0.05 is 2.0.

Id. A relative risk of 1.0 suggests that there is no association between the product and the disease, that is, the same numbers of people using the product are diagnosed with the disease as those not using the product. Similarly, a relative risk of less than 1.0 suggests that the product is actually "protective" of the disease: fewer people using the product contract the disease than those not taking the product. *Id.* at n. 5.

In general, epidemiology studies are probative of *general causation*: a relative risk greater than 1.0 means the product has the capacity to cause the disease. "Where the study properly accounts for potential confounding factors and concludes that exposure to the agent is what increases the probability of contracting the disease, the study has demonstrated general causation-that exposure to the agent is capable of causing [the illness at issue] in the general population." *Id.* at 893 (internal quotation marks and citation omitted).

In the Ninth Circuit, such studies can also be probative of *specific* causation, but only if the study shows the relative risk is greater than 2.0, that is, the product more than doubles the risk of getting the disease. *Daubert v. Merrell Dow Pharms. Inc.*, 43 F.3d 1311, 1315 (9th Cir.), *cert. denied* 516 U.S. 869, 116 S.Ct. 189, 133 L.Ed.2d 126 (1995) (" Daubert II"). When the relative risk is 2.0, the alleged cause is responsible for an equal number of cases of the disease as all other background causes present in the control group. Thus, a relative risk of 2.0 implies a 50% probability that the agent at issue was responsible for a particular individual's disease. This means that a relative risk that is greater than 2.0 permits the conclusion that the agent was more likely than not responsible for a particular individual's disease.

B. TREATING PHYSICIANS

Defendant's have moved this court to prohibit Henricksen's treating physicians from testifying on the subject of causation because they were not timely disclosed as experts on this subject and did not prepare expert reports.

John Caton, M.D. was one of Henricksen's treating physicians. He is board certified in hematology, internal medicine, and oncology. Although Mrs. Henricksen testified during her deposition that during a doctor visit Dr. Caton indicated Mr. Henricksen's AML was likely related to his occupational exposure to benzene contained in gasoline, Dr. Caton was disclosed only as a fact witness. Plaintiffs indicated he would testify "as to all aspects of the care and treatment of Mr. Henricksen, including diagnosis, treatments, prognosis, and the nature and course of the disease and its affects on human physiology." Ct. Rec. 44 [Plaintiffs' Final Trial Witness List]. Plaintiffs separately listed other expert witnesses who would address causation.

In early January, 2008, defense counsel inquired in emails whether the treating physicians would address causation. Plaintiffs counsel responded "I don't know. I have not hired them as experts and so I assume that they will say what they will." Ct. Rec. 70 (Weiss Decl.), Ex. 2 [Jan. 4, 2008 email].

None of Caton's chart notes contain an entry opining on causation. Dr. Caton did not prepare an expert report, which would have been due February 1, 2008.

Eight days before the discovery cutoff, on April 24, 2008, Plaintiffs revealed in an email to defense counsel that Dr. Caton would address causation. Dr. Caton was not deposed until after the discovery cutoff. Immediately prior to his deposition, Plaintiffs' lawyer gave Dr. Caton highlighted materials including an epidemiological article and a Conoco Material Safety Data Sheet (MSDS). Dr. Caton testified at his deposition that 1) common sense told him that Henricksen's AML was probably the result of having been exposed to benzene in the gasoline he hauled for decades; 2) that he had read in medical textbooks, including "Haskell" (which he claimed to have brought with him to the deposition) about the connection between gasoline and AML; and 3) the cause of Henricksen's disease played no role in Henricksen's diagnosis, prognosis or treatment.

Weiss Decl., Ex. 9 [Caton Depos.] at 176:7-10. Defense counsel had no advance notice of Caton's reliance upon "Haskell" as a source. After the deposition defense counsel located the text Haskell and it was discovered that the page at the deposition was not in that text and Haskell's chapter on AML makes no reference to gasoline.

Plaintiffs claim that no one talked to Dr. Caton until the end of the discovery period, and for that reason, no one knew what his testimony would be. Plaintiffs admit they did not "track[] down" Dr. Caton to ask him whether he held an opinion as to what caused Henricksen's AML until the end of the discovery period. Caton informed Plaintiffs' counsel at that time that it was caused by work around the benzene in the gasoline fumes, and that he had formed that opinion at the time of his diagnosis. Plaintiffs argue Dr. Caton is not subject to the reporting requirements of Fed. R.Civ.P. 26(a)(2)(B) because he isn't considered a "retained expert" because he's a treating physician, and causation is part of the ordinary care of a patient (and an observation Caton made at the time of his care).

Courts generally allow expert testimony by treating physicians without need for a report, provided that the opinions do not extend beyond treatment. *See Fielden v. CSX Transp. Inc.*, 482 F.3d 866, 870-71 (6th Cir. 2007) (holding that a formal report is not required when determining causation is an integral part of treating a patient); *see also Watson v. United States*, 485 F.3d 1100, 1107 (10th Cir. 2007). Nevertheless, even if a party elects to call a physician to testify solely to the treatment, the party must still, consistent with the court's scheduling order, disclose that person as someone he or she intends to call as an expert. *See Musser v. Gentiva Health Services*, 356 F.3d 751, 757-58 (7th Cir. 2004). "Disclosing a person as a witness and disclosing a person as an expert witness are two distinct acts." *Id*. The court held that "[f]ormal disclosure of experts is not pointless. Knowing the identify of the opponent's expert witnesses allows a party to properly prepare for trial. [The defendant] should not be made to assume that each witness disclosed by the [plaintiffs] could be an expert witness at trial." *Id*.

Dr. Caton's opinion on causation in this case is a type of expert testimony which

"results from a process of reasoning which can be mastered only by specialists in the field." Fed.R.Evid. 701, advisory committee's note, 2000 Amendments. Accordingly, Plaintiffs were required to have disclosed Caton as an expert witness under Rule 26(a)(2)(A). Rule 37(c)(1) "gives teeth to [this] requirement[ ]," *Yeti by Molly, Ltd. v. Deckers Outdoor Corp*., 259 F.3d 1101, 1106 (9th Cir. 2001), by prescribing: "If a party fails to provide information or identify a witness as required by Rule 26(a) ... the party is not allowed to use that ... witness to supply evidence ... at a trial, unless the failure was substantially justified or is harmless." Fed.R.Civ.P. 37(c)(1). The Advisory Committee Notes to the 1980 Amendment to Rule 37 state that this is an "automatic," "self-executing sanction." "[T]he burden is on the party facing sanctions to prove harmlessness." *Yeti by Molly*, 259 F.3d at 1106. The Ninth Circuit analyzed this harmless element in *Yeti by Molly* "by looking at whether the failure to disclose the information prejudiced the opposing party." *Galentine v. Holland Am. Line-Westours, Inc.*, 333 F.Supp.2d 991, 993 (W.D.Wash. 2004).

The need to indicate any treating physician that may be called as an expert is necessary for the defense to adequately prepare, and the Defendant will not be made to assume that such witnesses could be experts. *See Musser, supra*. Plaintiffs have not sustained their burden of showing Defendant would not be harmed if the late disclosure is allowed. Allowing Dr. Caton to be disclosed as an expert witness at this stage - well beyond the discovery cutoff - of the proceeding is not harmless. For the reasons stated, Defendant's Motion to Exclude Dr. Caton's Proposed Causation Testimony **(Ct. Rec. 67)** is **GRANTED**.

This ruling renders Defendant's *Daubert* motion to exclude Dr. Caton's opinions **(Ct. Rec. 127) MOOT**. However, even if the court were to disregard the disclosure violation, Dr. Caton's general and specific causation testimony would be subject to exclusion under *Daubert*, because his opinion lacks any reliable support. Dr. Caton himself admits that he is not rendering a "scientific opinion" and that he did not consult

any literature before opining that AML can be caused by gasoline exposure and that Henricksen's AML most likely resulted from his occupational exposure.

In addition to Dr. Caton, Henricksen was treated by Drs. Nichols, Georges, and Kominsky. The same reasons as justify the exclusion of Dr. Caton's causation opinion**,** would justify prohibiting these physicians from offering expert testimony. They were not disclosed by Plaintiffs as expert witnesses on any subject and Defendant would be materially prejudiced at this point, should Plaintiffs attempt to solicit expert testimony from them at trial. Defendant's motion to exclude any such testimony is unopposed as no response was filed by Plaintiffs. Accordingly, Defendant's Motion in Limine to Limit the Testimony of Plaintiffs' Treating Physicians **(Ct. Rec. 71)** is **GRANTED**.

C. FRANK GARDNER

Frank Gardner is a medical doctor from Galveston, Texas retained by the Plaintiffs for a causation opinion (both general and specific). Gardner rendered his report on November 7, 2007 wherein he opines Henricksen's "prolonged exposure to benzene in gasoline, initiated mutagenic changes in the bone marrow leading to his leukemia." Ct. Rec. 148 [Gardner Decl.], Ex. A. Gardner's methodology in reaching this conclusion is as Defendant describes: Henricksen has AML; Henricksen worked as a tanker truck driver loading and unloading gasoline; gasoline contains benzene; therefore Henricksen's AML was caused by exposure to gasoline. Gardner admits that gasoline is not identified by any regulatory body as a human carcinogen. Plaintiffs admit that Gardner did not have a specific dose calculation before reaching his opinion and that he relied upon the "universal knowledge that gasoline contains benzene and benzene causes AML at very low doses." Ct. Rec. 146 at 70.

Defendant asserts Rule 702 bars Dr. Gardner's testimony because his methodology for reaching his conclusions is flawed. Specifically, Defendant points out that (1) Gardner admits gasoline is not identified as a carcinogen; (2) Gardner fails to cite to any epidemiological study (or objective source) demonstrating that gasoline causes AML, he

merely asserts his personal opinion; (3) Gardner fails to consider any other possible causes of Henricksen's disease; (4) Gardner has no evidence of the dose of gasoline or benzene to which Henricksen was allegedly exposed and he did not attempt to ascertain actual exposure to either gasoline or benzene (Gardner Deposition at 59:17-20; 59:23-60; 49:5-9); and (5) Gardner relies upon incomplete or erroneous underlying facts including incorrectly assuming Henricksen's job duties involved loading 90% gasoline, that he stood above the hatch and sometimes on the ground while loading his truck, and that he kept his truck running while unloading it.

The Ninth Circuit requires general causation opinions to be supported by reliable epidemiological studies or if there are none, a reliable differential diagnosis through which, to a reasonable degree of medical certainty, all other possible causes of the victims' condition can be eliminated, leaving only the toxic substance as the cause. Gardner's opinion that exposure to benzene as a component of gasoline can cause AML is not supported by reliable studies and is instead based upon assumptions. In fact, Gardner testified in his deposition that he did not find it important or relevant to support his opinion by studies. Ct. Rec. 78, Ex. A (Gardner at 87:24-88:2). Gardner's inability to point to any source which reliably supports his conclusion, renders his opinion merely personal opinion. The scientific record does not reflect that there is "universal" knowledge that benzene causes AML at very low doses, as Gardner concludes. Because of this lack of scientific support, the court can not conclude Gardner's opinion is based upon scientifically valid principles, and therefore under Daubert, his general causation conclusion must be excluded.

Gardener's specific causation opinion also has severe problems. Though proof of Henricksen's benzene exposure through specific quantitative measurement is not a requirement, exposure at some level must be shown before a link between benzene exposure and AML could be drawn. All of the experts in this case agree benzene-induced diseases are dose dependent, and Gardner testified in his deposition that duration

and frequency of exposure are important factors to consider. Yet, in his report, Gardner did not attempt to quantify dose or even estimate Henricksen's level of exposure to benzene. Implicitly, Gardner's opinion presumes that exposure to benzene in gasoline can cause AML in any dose and that Henricksen's exposure was sufficient. Gardner's opinion is undermined by his failure to analyze or evaluate (his own or any other expert's) information pertaining to dose or the actual level of Henricksen's exposure. This renders his opinion on specific causation inherently unreliable. The court notes the shortcomings of Gardner's testimony in this case are similar to those identified by the *Castellow* court in a case very similar to this one, in which Gardner's opinion was excluded, *Castellow v. Chevron USA*, 97 F.Supp.2d 780, 795 (S.D. Tex. 2000).

Likewise, in order to result in an admissible conclusion, Gardner's analysis should "reliably rule out reasonable alternative causes of [the alleged harm] or idiopathic causes." *Soldo v. Sandoz Pharmaceuticals Corp*., 244 F.Supp.2d 434, 567 (W.D.Pa. 2003). Admissible expert testimony need not rule out all alternative causes, but "where a defendant points to a plausible alternative cause and the doctor offers no explanation for why he or she has concluded that it was not the sole cause, that doctor's methodology is unreliable." *Heller v. Shaw Indus., Inc.*, 167 F.3d 146, 156 (3d Cir.1999) (quoting *In re Paoli Railroad Yard PCB Litig*., 35 F.3d 717, 759 n. 27 (3d Cir. 1994).

Here, Gardner (and all of Plaintiffs experts, for that matter) fail to exclude-much less address in their reports-the likelihood that Henricksen's AML had no known cause. The affidavits of Pyatt and Natelson are unrebutted as to the fact that 80-90% of all cases of AML are idiopathic, having no known cause. Faced with similar situations, other courts have excluded experts' differential diagnoses where they failed to adequately account for the likelihood that the disease was caused by an unknown factor. *Doe v. Ortho-Clinical Diagnostics, Inc.*, 440 F.Supp.2d 465, 478 (M.D.N.C. 2006), for example, excluded the testimony of plaintiffs' expert because "he did not properly perform the differential diagnosis given his failure to consider within his analysis the high probability

that an unknown genetic cause cannot be ruled out as the specific cause of Minor Child Doe's autism." Similarly, in *Whiting v. Boston Edison Co.*, 891 F.Supp. 12 (D.Mass. 1995), the court excluded expert testimony that radiation was the cause of plaintiff's acute lymphocytic leukemia. The court reasoned that "[d]ifferential diagnosis, as the technique is used in the medical profession, consists of the comparison of a patient's symptoms to symptoms associated with a known set of diseases. The idea is to find the disease that matches the symptoms. If 90 percent of the causes of a disease are unknown, it is impossible to eliminate an unknown disease as the efficient cause of a patient's illness." *Id*. at 21 n. 41.

It seems the only reason cited for distinguishing Henricksen's disease from one of "no known cause" was the existence of a known risk factor, namely exposure to benzene. Standing alone, the presence of a known risk factor is not a sufficient basis for ruling out idiopathic origin in a particular case, particularly where most cases of the disease have no known cause. This is not to say that where most diagnoses of a disease are idiopathic it is impossible to prove specific causation. But in those cases, analysis beyond a differential diagnosis is required. Gardner could have compared the presentation of Henricksen's symptoms with those in chemically induced AML cases. However, in this case, perhaps they didn't because doing so would not have served Plaintiffs' purposes. Henricksen's presentation is very different from the typical case of chemically induced AML. None of the features characteristic or commonly seen in secondary AML have been associated with Henricksen's case. Thus in addition to the reasons cited above, because Gardner's methodology employed fails to adequately account for the possibility that Henricksen's AML was idiopathic, the court finds that his conclusion that prolonged exposure to benzene in gasoline was the cause of his AML is unreliable and therefore inadmissible.

D. WILLIAM SAWYER

1. DISCLOSURE

Defendant has moved to strike the testimony of William Sawyer arguing Plaintiffs

violated the rules of discovery in making his disclosure. William Sawyer was disclosed by Plaintiffs as a *toxicology* expert who would testify to the "toxic effects of benzene and gasoline, the mechanisms of injury, the medical literature, and general and specific causation in this case." Ct. Rec. 44 [Final Witness List filed 12/14/2008]. On the same disclosure, Plaintiffs identified Marco Kaltofen as an expert on *dose*, because this was the specific area on which he would opine. On February 1, 2008, Plaintiffs disclosed Sawyer's expert report in which he expressed opinions regarding dose. Sawyer was not deposed until March 13, 2008, at which time he was extensively questioned about his dose calculations. On April 21, 2008, eleven days before the discovery cutoff, Plaintiffs served a supplemental disclosure regarding Sawyer indicating they would have Sawyer testify to everything stated in his report and at his deposition.

Defendant's motion to strike argues that Sawyer's disclosure as a dose expert was untimely and that fairness and judicial economy justify striking his dose opinion based upon its late disclosure. Defendant contends that it selected just one expert on dose, relying upon the presumption that Plaintiffs also only had one expert on dose. Defendant's also contend Sawyer's dose testimony is cumulative because Plaintiffs already have a dose expert - Kaltofen.

The court finds that the Defendant should not have been "surprised" that any testimony related to toxicology could include opinions on dose, nor is it unusual to have two experts on dose. Plaintiffs timely disclosed Sawyer as an expert on both specific and general causation. Defendant was not prejudiced because it had already retained a dose expert who had time to review Sawyer's initial disclosure relating to dose in preparing his expert report. Moreover, Sawyer was deposed prior to the deposition of their dose expert. Accordingly, Defendant's Motion to Strike Dose Opinions of Sawyer due to Disclosure Violations **(Ct. Rec. 79)** is **DENIED**.

2. DAUBERT

Defendant's also move the court to exclude the opinions of Williams Sawyer and

preclude others from relying on his opinions, based upon *Daubert*. The court finds in Defendant's favor that Plaintiffs have not demonstrated that Sawyer's testimony is the product of reliable principles and methods, and that he has applied the principles and methods reliably to the facts of the case. At each step of his analysis, Sawyer bases his analysis upon speculation and/or erroneous data, and without adequate explanation, these steps render his methodology unreliable and misleading, and his opinions are therefore inadmissible.

Sawyer's opinion is that Henricksen was exposed to "dangerously high levels" of benzene which primarily contributed to his development of AML. Ct. Rec. 151, Ex. A. In essence, Sawyer's methodology to calculate Henricksen's dose was as follows. First, Sawyer estimated the amount of benzene Henricksen was likely exposed to on a daily basis. To do this he relied upon the baseline benzene exposure value reached in a study (*Kawai*[2]) of 10 Japanese truck drivers, under unknown conditions, during a 14-minute loading activity. The calculation from the Japanese study he relied upon for estimating Henricksen's dose was the arithmetic mean value (5.2 ppm, which includes outliers) as opposed to the geometric mean (2.89 ppm, which is the central tendency of the data). Sawyer then adjusted the number to fit Henricksen's work experience. Sawyer assumes Henricksen top-loaded gasoline at the Conoco terminal for 43% of his total work week, and then concludes that Henricksen was exposed to 5.2 ppm benzene for 43% of his work week. Sawyer then multiplies 5.2 ppm x 0.43 to arrive at 2.236 ppm, which he represents as Henricksen's exposure during his loading activities. Because Henricksen loaded gasoline only 50% of the time while at the Conoco terminal, this 2.236 ppm figure is reduced by half to yield Henricksen's annual benzene exposure estimate of 1.118 ppm, which Sawyer rounds up 1.12 ppm. Finally, Sawyer multiplies 1.12 ppm by the 8 years he says Henricksen worked at the Conoco terminal loading gasoline (1976-1984) to reach

[2] Kawai, T., et al. *Exposure to Vapors of Benzene and Other Aromatic Solvents in Tank Truck Loading and Delivery*, 46 BULL. ENVIRON. CONTAM. TOXICOL. 1-8 (1991).

a cumulative dose of 8.9 ppm-years (1.12 ppm x 8 years).

It is impossible not to be skeptical of both Plaintiffs' specific causation experts methodologies when their annual dose estimates diverge from one another by a factor of 471%. The scientific basis for Sawyer's reliance on *Kawai* for his baseline calculation is far from clear. Based upon Sawyer's review of Henricksen's testimony and historic photos of the enclosed nature of the terminals Henricksen worked at, Sawyer speculates that Henricksen's "exposures during top loading certainly fall within the high end of the published studies." Sawyer then just assumed that Henricksen's exposures were "at least" the 5.2 ppm calculated in the *Kawai* study. Ct. Rec. 151, Ex. A at 26. Sawyer states his reliance upon Kawai was due to the fact it involved top-loading gasoline which had similar benzene content (0.91% - 2.08%). None of the other conditions in which the workers in the *Kawai* study operated were known (e.g., closed versus open terminal, etc). There have been numerous large-scale studies in which actual benzene exposure measurements have been taken. Instead of relying upon available studies of U.S. fuel transport drivers using a full 8-hour shift, the 5.2 ppm baseline stems from a single study of just 10 Japanese truck drivers, under unknown conditions, and based upon only short-term 14 minute time intervals (involving only top-loading). As Defendant indicates, the accepted practice when performing a dose estimate is to apply a time-weighted value, not a short-term value. Ct. Rec. 85 at 6. Using short-term sampling data to extrapolate to long term exposures results in high rates of error. *Spencer*, ¶ 12. Even Sawyer himself recognized that a short-term task-based sample could not be accurately used to calculate a time-weighted average value (for an entire 8 hour shift), unless the person's only exposure occurred during that 14 minute interval. Ct. Rec. 86, Ex. A [Sawyer depos.] at 182:20-25. While Rule 702 does not require an expert to find a study linking the exact facts, it does require the expert demonstrate a scientifically valid basis for projecting the findings of a study to the proffered casual theory. Sawyer has not provided an adequate basis for reliably linking the values derived from the circumstances of *Kawai* to the

circumstances of Henricksen's case.

Sawyer's use of 5.2 ppm from the *Kawai* study also is problematic because this value represents the arithmetic mean (which includes outliers) as opposed to the geometric mean (2.89 ppm - which is the central tendency of the data) for estimating Henricksen's dose. Generally accepted methodology would use the geometric mean. Ct. Rec. Ex. , *Spencer*, ¶ 6 (citing scientific papers). Sawyer also admitted during his deposition that geometric mean value would better estimate the central values of the exposures of the Japanese truck drivers. Using the geometric mean of the already small sample instead of the arithmetic mean would reduce Sawyer's dose calculation by approximately half. When asked at his deposition whether he'd like to correct his calculation from 8.9 ppm to 4.8 ppm cumulative benzene exposure to compensate for this, Sawyer indicated that "either of those numbers are actually valid." Sawyer Depos. at 283:8-14.

Sawyer's speculation in estimating Henricksen's baseline exposure and the resulting widely varying calculations which might be correct or might not be correct, make Dr. Sawyer's opinion confusing and unreliable. Adding to the confusion, as Defendant points out, Sawyer's adjustments to account for Henricksen's work environment tended to overestimate the actual facts. For example, Henricksen's testimony would result in Henricksen hauling a mean of 21 loads per 6-day work week, rather than the 27.5 loads assumed by Sawyer (based upon an erroneous calculation of counsel during a deposition). Sawyer created the time weighted average using a 40 hour work week, instead of a 55-60 hour work week, which Henricksen also testified he usually worked. Finally, Sawyer estimated the time Henricksen transported gasoline as 8 years, instead of the more accurate figure of 7.548 years. Taken individually, the court agrees with Plaintiffs, Sawyer's use of these figures is not so egregious as to undermine his analysis and could be readily pointed out on cross-examination or corrected. However, cumulatively, these errors enhance the court's initial conclusion based upon the

unreliable reliance on *Kawai*, that Dr. Sawyer's lacks precision and is unreliable science.

Sawyer's statement that either calculation - 8.9 or 4.8 ppm-years - would be valid (and would not change his final conclusion), gives insight into Sawyer's model for his opinion. As stated in his report dated March 10, 2008, he states that he believes the quantified dose of exposure "takes on far less significance" in a cancer case, because when no safe-threshold of exposure to a carcinogen has been established, each and every exposure will increase the development of cancer. Given the fact that humans are exposed to background levels of carcinogens on a daily basis, in his view, causation evidence of exposure to the carcinogen must be "more than trivial, hypothetical, negligible or theoretical." Ct. Rec. 151, Ex. B at 23. "[E]ven very small exposures to carcinogens *should* be considered substantial risk factors for cancer..." *Id*. at 24 (emphasis added). This reasoning explains why Sawyer would assign benzene as a component of gasoline as a cause of Henricksen's AML regardless of whether calculated dose was 8 or 4, so long as his dose was something more than negligible.

The use of the no safe level or linear “no threshold” model for showing unreasonable risk “flies in the face of the toxicological law of dose-response, that is, that ‘the dose makes the poison,’ which refers to the general tendency for a greater dose of a toxin to cause greater severity of responses in individuals, as well as greater frequency of response in populations.” Federal Judicial Center, Reference Manual on Scientific Evidence 475 (2d ed. 2000). Other courts have similarly rejected expert opinions that are based on the “no-threshold” model. As one court explained in excluding the plaintiffs' experts using the same no threshold theory, “[t]he linear non-threshold model cannot be falsified, nor can it be validated. To the extent that it has been subjected to peer review and publication, it has been rejected by the overwhelming majority of the scientific community. It has no known or potential rate of error. It is merely an hypothesis.” *Whiting v. Boston Edison Co.*, 891 F.Supp. 12, 25 (D.Mass. 1995). “In layman's terms, the model assumes that if a lot of something is bad for you, a little of the same thing,

while perhaps not equally bad, must be so in some degree. The model rejects the idea that there might be a threshold at which the neutral or benign effects of a substance become toxic." *Id.* at 23. *Sutera v. Perrier Group of America Inc*., 986 F.Supp. 655, 666 (D.Mass.1997) ("Accordingly, although there is evidence that one camp of scientists ... believes that a non-linear model is appropriate basis for predicting the risks of low-level exposures to benzene, there is no scientific evidence that the linear no-safe threshold analysis is an acceptable scientific technique used by experts in determining causation in an individual instance.").

Sawyer's theory that any amount of exposure more than negligible should be considered substantial risk factor for AML flies in the face of the scientific literature reviewed and other expert testimony in this case that there is a threshold or dose below which you do not see a statistically significant risk of developing AML. Ct. Rec. 104, Ex. A [Gardner Depos.] at 70:3-15. Even though benzene has been shown to cause AML, it is too difficult a leap to allow testimony that says any amount of exposure (above the short term exposure limits) to this toxin can cause AML and caused AML in Henricksen.

Because Sawyer's dose calculation is unreliable and his belief that low-dose exposure to benzene as a component of gasoline is capable of causing AML a hypothesis rather than science sufficiently reliable for causation purposes, it is fails the *Daubert* factors. Accordingly, Defendant's motion to exclude the testimony and opinions of Plaintiff's Expert, William Sawyer, (Ct. Rec. 83) is **GRANTED.**

E. MARCO KALTOFEN

Defendant submits a Motion in Limine to Exclude the Testimony of Plaintiffs' Expert Witness Marco Kaltofen (Ct. Rec. 87). Kaltofen was retained by Plaintiffs to calculate dose for specific causation. Kaltofen's methodology for calculating dose begins by estimating a value of 0.38 ppm to estimate Henricksen's "unadjusted daily benzene

exposure." He has cited to two studies for support for this figure, *Irving & Grumbles*[3] (in his report) and (*Verma*[4]) (at his deposition). From this baseline, Kaltofen adjusts for the fact that Henricksen only loaded gasoline 50% of the time (as opposed to the drivers in *Verma* who loaded 80% of the time), coming up with 0.2375 ppm-years for Henricksen's annual exposure. He multiplies this by 7 years he loaded at the Conoco terminal (1976-1983), resulting in a cumulative dose estimate of 1.6625 ppm-years. He then multiplies that number by a factor of 5 to accommodate the fact that Henricksen worked at a terminal with a roof to come up with a cumulative dose estimate of 8.3125 ppm-years.

Defendant's motion maintains Kaltofen's opinions, reports and testimony should be excluded under Rule 702 because his estimate is scientifically unreliable. In its accompanying memorandum, the Defendant elaborates on its objections by assigning two primary defects to his testimony. First, Defendant takes issue primarily with Kaltofen's 5x multiplier and reliance upon the *Nordlinder*[5] study. Defendant contends Kaltofen's 5x multiplier has never been tested, peer-reviewed, and no scientific literature, treatise or professional association has ever recognized its existence, or reliability. *Nordlinder*, Defendant's contend, does not discuss a multiplier nor stand as a reliable study of the difference between exposures open and closed terminals. Second, Defendants take issue with Kaltofen's baseline number, arguing he relies erroneously upon a value which was described as the permissible occupational exposure limit, rather than any actual exposure

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[3] Irving, W.S. and Grumbles, T.G., *Benzene Exposures During Gasoline Loading at Bulk Marketing Terminals*, 40 AMER. INDUSTRIAL HYGIENE ASSOC. JOURN. 468 (1979).

[4] Verma, D.K., et al., *A Simultaneous Job- and Task-Based Exposure Evaluation of Petroleum Tanker Drivers to Benzene and Total Hydrocarbons*, 1 JOURN. OF OCCUP. AND ENVIRON. HYGIENE 725 (2004).

[5] Ct. Rec. 90, Ex. B [Nordlinder, Rolf et al, *Exposure to Benzene at Different Work Places in Sweden*, 31 J. OF OCC. HYGIENE 345 (1987)

measurement (which would have had a central value of 0.365, instead of .38).

The court gives most credence to Defendant's objection to Kaltofen's reliance upon *Nordlinder* for the 5x multiplier. Nordlinder is a study which involved an investigation of benzene exposures during loading operations at two Swedish fuel terminals. The study describes in table form the results of the study. Measurements were obtained for five workers at a "closed terminal building" and for 16 workers at an "open terminal building." The mean benzene concentration for open terminals was 4.3 units, whereas the mean benzene concentration for closed terminals was 22.0 units. The study does not describe the difference between open and closed terminals, although the study notes terminals with "less effective natural ventilation" showed higher levels of benzene concentrations.

Because the difference between *Nordlinder's* average values for open and closed terminals was approximately five times, Kaltofen concluded he should multiply Henricksen's dose by a factor of five because the terminal he operated in had a shed/roof over it. However, the *Nordlinder* study does not suggest that a multiplier should be derived from its data, nor does the author suggest it intended its study as a comparison of open versus closed terminals. When Kaltofen wrote his report he had no information on what constituted a "closed" or "open" terminal in Sweden. Without explanation, he simply hypothesizes without foundation that Henricksen's occupational circumstances (a terminal with a roof and two partial walls) can be equated to the closed terminals in Sweden and this study involving only five workers. Kaltofen's five time multiplier derived from a study has not been tested and is not recognized by any other expert or in any other literature provided to the court. The fact that the multiplier was crafted from this study by Kaltofen for purposes of rendering an opinion in this litigation, and the failure of Kaltofen's methodology to have been peer reviewed or otherwise tested, weighs strongly against admitting the testimony.

The court also considers the potential rate of error. *Nordlinder* was not an

appropriately designed study to yield reliable or conclusive results on the difference between benzene exposures in open and closed terminals. The small sample sizes of five and sixteen leaves a great deal of uncertainty about the measurements obtained. If in error, Henricksen's cumulative dose calculation could be off by 500%. Kaltofen's methodology in arriving at the multiplier of 5 shows a lack of scientific rigor in that he expands the application of *Nordlinder* beyond good science, drawing conclusions the authors of the study did not make from limited data. It is this kind of scientifically unsupported "leap of faith" which is condemned by *Daubert*. *See Rink v. Cheminova, Inc.*, 400 F.3d 1286, 1292 (11th Cir. 2005) (excluding expert testimony because experts method of transposing data from other studies based on such conjecture and rough approximation lacked the "intellectual rigor" required by *Daubert*). Accordingly, Kaltofen's dose opinion must be excluded and Defendant's motion (Ct. Rec. 87) **GRANTED**.

F. PETER INFANTE AND THE EPIDEMIOLOGICAL EVIDENCE

Defendant's have filed a separate *Daubert* motion to exclude the testimony of all the Plaintiffs' general causation experts, including Dr. Peter Infante, Sawyer, and Gardner, based upon their reliance upon allegedly unreliable or irrelevant epidemiological studies. Absent definitive support for the theory that gasoline can cause AML, Defendant contends Plaintiffs must support their opinions by "statistical epidemiological evidence." *Hanford*, 292 F.3d at 1136. In Defendant's view, Plaintiffs "want to expand the frontiers of science through the courtroom to find something that science itself has rejected." According to the Defendant, there are no reliable studies that show that the benzene contained in gasoline can cause AML at the exposures Henricksen is alleged to have had.

After considering the proffers of testimony in light of the prevailing case law, the court finds that it must exclude Dr. Infante, Sawyer and Gardner's general causation testimony because to the extent that the data relied upon is itself scientifically reliable, it

does not support the proffered causation conclusion since there is “too great an analytical gap between the data and the opinion proffered.” Furthermore, the court can not permit Dr. Infante's specific causation testimony in the absence of any admissible evidence of general causation and because Kaltofen's dose calculation he relied upon is inadmissible.

Peter Infante is Plaintiffs’ expert epidemiologist who opines both on general and specific causation. In his first report dated November 29, 2007 (Ct. Rec. 149, Ex A) he conducts a review of various epidemiological studies and opines that:

(1) Occupational exposure to benzene is associated with AML;

(2) Some epidemiological studies of occupational exposure to gasoline containing 1-2% benzene demonstrate an elevated risk of leukemia, including AML, with cumulative benzene exposures of as low as 1.5 ppm-years;

(3) Epidemiological studies demonstrate "significantly elevated risks" of AML for among those who transport gasoline and those who are engaged in terminal work at refineries where gasoline is loaded and unloaded;

(4) There is no evidence that *cumulative* benzene dose has been validated as the proper metric for assessing whether benzene causes leukemia (Ct. Rec. 149 at 14); and

(5) The genotoxic effects of benzene in gasoline support the biological plausibility of gasoline to induce leukemia.

In his second report, dated February 1, 2008, Dr. Infante gives his general and specific causation opinions. He opines that "epidemiological studies demonstrate **significant associations** between gasoline exposure and the development of AML" and "the literature demonstrates **the same** toxic effects for gasoline as seen for benzene." Finally, he concludes, "[b]ased upon my review and analysis of the data related to Mr. Henricksen's employment and my review and analysis of the scientific literature related to the toxicity of benzene, gasoline exposure, and to other causes of AML, it is my opinion that Mr. Henricksen's 29 years of occupational exposure to gasoline was a substantial contributing factor and the most likely cause of his development of AML."

Ct. Rec. 149, Ex. B (emphasis added).

The record evidence proffered by the parties on these motions evidences their understanding of the particularly acute need for epidemiology in the instant case. Plaintiffs' experts have not rebutted the showing that AML occurs in the general population. Nor did they rebut the showing by Defendant that 80-90% of all AML cases have no known cause. Therefore, to determine whether any given case of AML could possibly be attributable to a particular toxin, epidemiology would be the favored methodology for scientifically testing the hypothesis that exposure to the toxin increases the risk of AML.

Defendant argues the scientific evidence upon which the Plaintiffs' experts rely does not qualify as reliable science or adequately support the contention that exposure to the benzene component of gasoline at the levels alleged is capable of causing AML. Defendant argues these inadequacies provide a basis under *Daubert* to exclude the expert testimony in this case and furthermore, even if they pass scrutiny under *Daubert*, the evidence is insufficient as a matter of law to withstand summary judgment on both general and specific causation.

Under the relevance prong of *Daubert*, the court must ensure that the proposed expert testimony logically advances a material aspect of the case. *Norris v. Baxter Healthcare Corp*., 397 F.3d 878, 884 n. 2 (10th Cir. 2005). *Daubert* and Fed.R.Evid 702 give the district court broad discretion to determine whether a body of evidence relied upon by an expert is sufficient to support that expert's opinion. Nothing in *Daubert* or the Federal Rules of Evidence requires a district court to admit opinion evidence that is connected to existing data only by the *ipse dixit* of the expert. *General Elec. Co. v. Joiner*, 522 U.S. 136, 146, 118 S.Ct. 512, 139 L.Ed.2d 508 (1997). At the same time, this court understands that in epidemiology hardly any study is ever conclusive, and the court does not suggest that an expert must back his or her opinion with published studies that unequivocally support his or her conclusions. *See Bonner v. ISP Techs., Inc*., 259

F.3d 924, 929 (8th Cir. 2001) (observing that "there is no requirement 'that a medical expert must always cite published studies on general causation in order to reliably conclude that a particular object caused a particular illness' ") (*quoting Heller v. Shaw Indus., Inc*., 167 F.3d 146, 155 (3d Cir.1999)).

To understand and evaluate the expert testimony, this court has engaged in a time consuming, intense evaluation of the expert reports and the voluminous scientific evidence which has been proffered in the form of exhibits to the court. The science on this matter is complex and evolving, and despite the steadfast positions of the parties expressed in this litigation, there continues to be considerable uncertainty in the scientific community regarding the link between low dose exposures to benzene as a component of gasoline and leukemia, specifically AML. This uncertainty is recognized throughout the literature. While absolute certainty is not what the law requires, expert opinions "must be based on facts which enable [the expert] to express a reasonably accurate conclusion as opposed to conjecture or speculation." *Jones v. Otis Elevator Co.*, 861 F.2d 655, 662 (11th Cir. 1988).

The court concludes herein that there is simply too great an analytical gap between the data presented and the opinions offered - especially as to specific causation - such that it renders the expert testimony too speculative as a matter of law. The court can not possibly exhaustively cover in this opinion each and every nuance of every study cited and debated by the parties. Thus, only the studies which are most central to the expert opinions and causation in this case are discussed.

Undeniably, the concern prompting the increasing number of studies of gasoline is the fact that it contains benzene, a toxin proven to cause AML. There have been numerous studies investigating the hazards of benzene (mostly as a component of other products) and also numerous studies with the focus being gasoline or petroleum products. Since it is the Plaintiffs' theory that low-dose exposure to the benzene in gasoline is capable of causing AML, one of the questions specifically raised in this litigation is at

what level of exposure the risks of AML become significant enough to suggest a causal relationship. The parties do not dispute studies have suggested cumulative *benzene* exposure of as low as 40-50 ppm-years significantly increases the risk of AML. One study published in 1995 of a cohort of workers employed at two Goodyear plants in Ohio in the manufacture of Pliofilm (rubber hydrochloride), showed no increase of AML was detected where estimated cumulative exposure was below 200 ppm-years (standard mortality rate (SMR)= 0.91). But above 200 ppm-years, the AML risk rose drastically (SMR 27.721 for 200-400 ppm-years and SMR 98.37 for >400 ppm-years). Ct. Rec. 104, Ex. I [Wong, Otto, *Risk of Acute Myeloid Leukemia and Multiple Myeloma in Workers Exposed to Benzene*, 52 OCCUP. AND ENVIRON. MED 380 (1995)]. Also worth noting, the study concluded it was important to account for specificity of disease in such studies because studies grouping all leukemia together rather than distinguishing by cell types could lead to inaccurate estimates of the actual risk for one or the other cell type.

It is Plaintiffs' theory that exposures of 8 ppm-years or lower could cause AML. Plaintiffs cite as particularly important to their theory that low dose exposure to benzene can cause AML, research regarding exposures to gasoline/benzene not in an occupational setting, but in a community setting. Reports created by the Pennsylvania Department of Health ("PADOH") regarding the Tranguch gasoline spill covered a cancer incidence study in the neighborhoods near the location where thousands of gallons of gasoline had leaked from underground storage tanks of several service stations and an auto repair garage. PADOH published reports in December 2001 (Ct. Rec. 163, Ex. 19) and an updated report in December 2003 (Ct. Rec. 163, Ex. 20). These ecological reports were not confined to the study of AML. The first report indicated the discovery of four incidences of leukemia (2 cases of AML) with standard incidence ratio (SIR)[6] which was

[6] Relative risk is a commonly used approach for expressing the association between an agent and disease, and is defined as the ratio of the incidence rate of disease in exposed individuals to the incidence rate in unexposed individuals. The incidence rate

deemed not statistically significant (2.36). The reports states the following in its concluding paragraphs:

> "..the types and proportion of cancers seen among residents of the spill site are essentially the same as seen for Pennsylvania....
> The relationship of leukemia incidence to the environment is not clear. Little is known about the factors responsible for the development of most types of leukemia. And only in rare circumstances can one say with certainty that an occurrence is due to a specific agent...
> Mechanisms for the induction of cancer from benzene exposure are not as clear...Demonstrated excess cancer risks from certain environmental exposures, including benzene have consistently come from very high exposure situations, such as industrial work sites. However, it has not ben possible to measure elevated rates in humans at much lower levels. This creates a situation where it is impossible to prove that ambient benzene levels are sufficient to cause cancer in the study population. Further characterization of environmental and other potential factors, for residents of Tranguch diagnosed with certain types of cancer, would be useful for evaluating risk.

Ct. Rec. 163, Ex. 19. PADOH'S updated report covering the period of 1985-2002 reported three additional cases of leukemia, one being AML. This made the risk ratio 5.56 (3 cases versus .054 expected), which the study concluded would leave the risk of occurring by chance sufficiently small to consider the increase in rate of AML statistically significant. Neither report drew any conclusions regarding the causal relationship between gasoline (or its benzene component) and leukemia or AML.

The University of Pittsburgh was also contracted by local officials to study the possibility of health effects on the Tranguch community. Patel. AS, et al., *Risk of Cancer As a Result of Community Exposure to Gasoline Vapors*, 59 ARCH. ENRIVON. HEALTH 497 (2004)(see Ct. Rec. 147, Ex. 42); *Patel A*, et al, *Chronic Low Level Exposure to*

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of disease reflects the number of cases of disease that develop during a specified period of time divided by the number of persons in the cohort under study. Federal Judicial Center Reference Manual on Scientific Evidence at 348 (2nd ed.2000)(hereinafter referred to as "Reference Manual"). If the relative risk is greater than 1.0, the risk in exposed individuals is greater than the risk in unexposed individuals; in other words, there is a positive association between exposure to the agent and the disease, which could be causal.

Gasoline Vapors and Risk of Cancer: A Community-based Study, 15 EPIDEMIOLOGY S192 (2004) (Ct. Rec. 104, Ex. N). This study followed the community for 7 fewer years, finding only 4 cases of leukemia (only 2 were AML). The incidence of all four cases of leukemia as opposed to the 0.91 expected (SIR = 4.40) resulted in what the authors considered a rate of leukemia "significantly higher" than state incidence. Notably, the authors concluded: "This research provides *potential* evidence of an increase in hematopoietic cancer risk as a result of longer-term exposure to low concentrations of benzene....Although an increase in the risk of leukemia as a result of exposure to low-level benzene remains equivocal, our study provides support for the growing body of evidence suggesting a causal link." Ct. Rec. 147, Ex. 42. The authors likewise concluded that "causality in this study cannot be determined since comprehensive historical exposure estimates were not available for individual residents." Ct. Rec. 104, Ex. N.

In 1993, the Agency for Toxic Substances and Disease Registry (ATSDR), part of the U.S. Department of Health and Human Services, also conducted a limited health consultation on the Tranguch site regarding the question of whether concentrations of benzene vapor were sufficiently high to have posed a public health concern. ATSDR, Health Consultation - Tranguch Gasoline Site, Hazelton, Luzerne County, Pennsylvania (March 17, 2004)(Ct. Rec. 104, Ex. O). Since the actual benzene levels and the duration and frequency of exposure were unknown, the ATSDR took a limited number of indoor air measurements over ten weeks, then based on a number of presumptions, *estimated* benzene exposure at approximately 2 ppm-years, with average exposures of 0.03 ppm-years. The authors admitted their data contained "much uncertainty" and noted its "[l]imitations...do not allow meaningful estimates of long-term cumulative exposure." The authors comment regarding their impression of the state of the epidemiological evidence at that time: "Exposure to benzene has been estimated from these studies to increase the risk of leukemia at the level of 40 ppm-years of cumulative exposure" and

"The lowest level of cumulative exposure associated with an increased incidence of acute nonlymphoctic leukemia among occupationally exposed workers has been in the range of 10-25 ppm." The authors draw no conclusions regarding causation, but recommend further investigation.

One of the primary benzene studies in an occupational setting Plaintiffs have relied upon is one study publishing the results of the Australian Institute of Petroleum's medical surveillance program called "Health Watch." This program consists of both a cohort study of the long-term health of oil industry employees and a case control study focusing on bone marrow cancers relationship to benzene exposure. Plaintiffs rely upon Glass, et al., *Leukemia Risk Associated with Low-Level Benzene Exposure*, 14(5) EPIDEMIOLOGY (Sept. 2003). *Glass* reports on a case-control study nested within the Health Watch cohort with subjects who were male petroleum workers with exposure to benzene in petroleum, as well as benzene concentrate (100%) and BTX, which contains approximately 70% benzene. The article itself notes that "[t]he extent of the risk of leukemia with exposure to low concentrations of benzene (less than 10 ppm) has been debated." The results of the study showed for cumulative benzene exposure in the category ">8" (ranging from 7.88 to 57.31 ppm-years), the odds ratio was 7.17 (CI =1.27-40.4). The conclusions the study's authors draw are that there is a strong association between benzene exposure and the risk of acute leukemia (which is undisputed by the parties in this case) and the excess risk of leukemia was associated with lower cumulative exposures and lower exposure intensity than have been observed in other studies. However, data on the actual exposures or jobs of the AML subjects in the study was not provided, thus it is impossible to say where on the ">8 ppm-years" continuum their exposures fell.

In November 2007, the thirteenth and most current Health Watch report (Ct. Rec. 104, Ex. U) was released (which is not cited by Dr. Infante). It reports there was no excess of AML in the Health Watch cohort in comparison with the general population.

The report notes that because of the differences between the *Glass* case control study and two major case-control studies of petroleum workers in the UK and Canada (discussed below), a separate study has been commissioned which will pool and update the data from all three studies. It is expected to be complete this year.

Of the studies Plaintiffs cite, the most relevant are those which address occupational gasoline exposure and AML. Three epidemiological studies which relate to occupational gasoline exposure and AML are relied upon. The most recent of those cited is Terry, Paul, et al, *Occupation, Hobbies, and Acute Leukemia in Adults,* 29 LEUKEMIA RES 1117 (2005). *Terry* was a case-control study involving surveys of 811 cases of adult acute leukemia (624 with AML) and 637 controls in the United States and Canada, who were interviewed by telephone. *Terry* states that "associations between certain occupational exposures and risk of AML...remain unclear." Twenty-seven different occupations were identified. Of the occupations that generally involve exposure to petroleum products, the study found a statistically significant association between AML risk and employment in automobile manufacturing, gas station attendant, and petroleum manufacturing. The study did not find such an association with automobile mechanics and truck, bus or taxi drivers. The study indicates that gas station attendant studies have tended to show positive associations (though there are some inconsistent studies), that gasoline contains benzene, but that benzene has been associated with the risk of AML "most clearly at levels above a threshold of 200 ppm-years."

Plaintiffs also cite to Flodin, U, et al, *Acute Myeloid Leukemia and Background Radiation in an Expanded Case-referent Study,* 45 ARCH ENV HEALTH 364 (1990). This was not a gasoline study. Rather *Flodin* was a study of the association of AML with electrical work and background radiation levels. Gasoline was noted by the authors as a confounder or "risk factor" (odds ratio 2.7 with a confidence interval of 90%), but the study was not statistically significant at the generally accepted confidence level of 95%.

Plaintiffs also rely upon Jakobsson for the proposition that occupational exposure to gasoline is linked to AML. Jakobsson, R., et al, *Acute Myeloid Leukemia Among*

Petrol Station Attendants, 48 ARCH. ENVIRON. HEALTH 255 (1993). *Jakobsson* studied the risk of AML within different occupations by sending questionnaires and conducting follow up interviews. The only occupation studied resulting in an increased risk of AML were "male petrol station attendants and demonstrators." Ten cases of AML were observed, yielding an odds ratio of 3.6. The authors contend, "A reasonable hypothesis was that exposure to benzene from petrol had contributed to the excess risk of AML, given that petrol in Sweden had contained up to 5% of benzene for several decades." Yet, the author admits that the lowest benzene air level that have been associated with increased risk of AML (40-199 ppm-years) are considerably higher than the levels measured at Swedish petrol stations. This study has been criticized for various reasons by other scientists, including for the discovery that 3 of the reported AML cases never worked as petrol service attendants and three others only did so for a short time. Ct. Rec. 94 (Garabrant Aff.).

As Infante (and Pyatt) indicate in their testimony, the gasoline station attendant studies have yielded inconsistent results. All of the experts have pointed out the single largest study of service station workers, *Lynge* et al (1997) (which is not part of the record), which showed there was no statistically significant increased risk of AML among the 19,000 gasoline attendants from the nordic countries (SIR 1.4, 95% CI 0.8-2.4).

There have also been numerous studies evaluating the risks of AML or leukemia associated with being an auto mechanic. Though Dr. Infante points out one of the studies (*Hunting* et al. (1995)) with a positive finding (SMR = 9.29 (95% CI = 1.12-33.43), Plaintiffs expert (*Pyatt* at13) states that at least 9 other studies do not report an increased risk associated with being an auto mechanic.

The experts agree that perhaps the most relevant studies to this case are those studying tanker truck drivers or gasoline distribution workers. Three major epidemiological studies involving very large cohorts of distribution workers have been conducted in Britain, Canada, and the United States. Each showed no statistical significance in the increase in the risk of AML among tanker truck drivers, though

nonsignficant excesses above 1.0 were found. Rushton, L., et al., *A 39-Year Follow-up of the United Kingdom Oil Refinery and Distribution Center Studies: Results for Kidney Cancer and Leukemia* 101 ENV. HEALTH PERSPECTIVES 77 (1993); Otto Wong et al., *Health Effects of Gasoline Exposure. II. Mortality Patterns of Distribution Workers in the United States*, 101 ENV. HEALTH PERSPECTIVES SUPPLEMENTS 63 (1993); Schnatter, A.R., *A Retrospective Mortality Study Among Canadian Petroleum Marketing and Distribution Workers*, 101 ENV. HEALTH PERSPECTIVES 85 (1993).

Schnatter (1993) was a retrospective mortality study of 6672 petroleum distribution workers in Canada but also involved separate examinations of those workers the study classified as "exposed to hydrocarbons" and the sub category of workers who had ever worked as a tank truck driver (1453 individuals). Ct. Rec. 147, Ex. 34. The leukemia findings (myeloid and lymphoid cell-types were grouped together) for the overall cohort and for those classified as "exposed to hydrocarbons" were not statistically significant. For the group of tanker truck drivers and the combined category of lymphatic and myeloid leukemias, the study showed a statistically significant standardized mortality ratio (SMR) of 3.87, based upon 4 cases. At that time, the authors could only confirm one definite case of AML. The study pointed out that it did not collect information on other risk factors (radiation, genetic predisposition, smoking) and that "[n]early all other analyses suggested the leukemia risk was not related to HC [hydrocarbon] exposure." In fact, the SMR was higher for the non-exposed group. The study does not draw any conclusions regarding biological feasibility of causation due to exposure to benzene acknowledging "[a]t this point we have no knowledge concerning the actual levels of benzene experienced by these truck drivers." Finally, the study concludes "...most of these results should be considered hypothesis generating and the impetus for future studies in these occupational groups."

Plaintiffs also cite to a review of these three studies, Enterline, P.E., *Review of New Evidence Regarding the Relationship of Gasoline Exposure to Kidney Cancer and Leukemia*, 101 (suppl. 6) ENVIRON. HEALTH PERSP. 101 (1993). Ct. Rec. 163, Ex. 37.

Enterline analyzes the studies and espouses that there is evidence for an AML relationship and that "it is possible that it is due to the benzene content of gasoline." He also writes "There was however, no trend in AML, when data were analyzed by various gasoline exposure indexes. While it is possible that the excesses are related to the benzene content of gasoline, they need further study in light of actual benzene exposure levels."

The results of *Schnatter's* study of the Canadian cohort were updated in 1996 (which notably, Dr. Infante did not cite in his report). In 1996, there was no statistically significant excess (SMR 1.09) of AML amongst tank truck drivers. Ct. Rec. 94, Ex. G *[Pyatt Aff.]*, at 11-12. Similarly, according to Pyatt, the updates of the other two large studies (mentioned above) of distribution workers in the U.S. (*Wong* et al (1999) and Britain (*Sorhan* 2002*)*, also reported no statistically significant relationship among truck drivers. *Pyatt* at 11-12.

In Sawyer's report he erroneously cites Lindquist, R. et al, *Acute Leukemia in Professional Drivers Exposed to Gasoline and Diesel*, 47 EUROPEAN J. OF HAEMATOLOGY 98 (1991), as a study of tankers and imprecisely infers it is an AML study (Table 1 in March 10, 2008 report). *Lindquist* is a study of all forms of acute leukemia (not just AML) and did not involve tanker truck drivers. Ct. Rec 94, Ex S. The study showed an increased risk among "professional drivers" for developing acute leukemia (odds ratio 3.0, 95% CI = 1.1-9.2). For those exposed for more than five years during their lifetime, the odds ratio was even greater. The court agrees that this study would suggest a relationship between acute leukemia for "professional drivers." Service station attendants and car mechanics did not have an increased risk of acute leukemia in this study. This study is at odds with other studies cited of drivers and while it does not really help answer the questions in this case, it is not wholly irrelevant.

In deciding whether the Plaintiffs' expert opinions have adequate scientific foundation, the court also has also considered the additional evidence proffered by the defense and defense experts. While reading what the authors of the epidemiological

studies actually say is most vital and revealing, the court notes that a large number of sources including the International Agency on Research on Cancer (IARC), the U.S. EPA, OSHA, and the Agency for Toxic Substances and Disease Registry have also reviewed this body of literature and published statements concluding that there is inadequate evidence to conclude gasoline is a carcinogen. The most current source in evidence is not a study, but an article, Jamall, I. and Willhite, C., *Is Benzene Exposure from Gasoline Carcinogenic*, 10 J. ENVIRON. MONIT.176 (2008). Its authors conduct an in depth review of the toxicological and epidemiological body of evidence on gasoline and benzene and they also conclude that "the majority of epidemiological studies conducted in various countries show no significant increase in the risk of leukemia, or where reported specifically, of AML." The article's authors remind the reader that "rigorous science looks for consistency between studies, biologic plausibility and most importantly, a dose-response relationship in the establishment of causality."

This brings one back to *Daubert*' s object with respect to the “reliability” prong: “to make sure that when scientists testify in court they adhere to the same standards of intellectual rigor that are demanded in their professional work. If they do, their evidence (provided of course that it is relevant to some issue in the case) is admissible even if the particular methods they have used in arriving at their opinion are not yet accepted as canonical in their branch of the scientific community. If they do not, their evidence is inadmissible no matter how imposing their credentials.” *Rosen v. Ciba-Geigy Corp.*, 78 F.3d 316, 318-19 (7th Cir.) (citations omitted), cert. denied, 519 U.S. 819, 117 S.Ct. 73, 136 L.Ed.2d 33 (1996).

The court concludes that Plaintiffs' experts' general causation testimony must be excluded because the studies they rely upon singly or in combination, do not support the causation conclusions they make in the face of the overwhelming body of contradictory and inconsistent epidemiological evidence. Plaintiffs experts have highlighted studies with positive associations - anything above 1.0, without regard to statistical significance - and from these associations concluded that the benzene in gasoline is capable of causing

AML. Actually, Dr. Infante did not even go so far as to say it was capable of causing AML, only that there are "significant associations" between gasoline exposure and the development of AML. However an association does not equal causation, and it is the duty of scientists to rigorously analyze the data to determine whether or not an association is causal. This means considering such factors as strength of association, consistency of association, specificity of the association, and biological plausibility.

None of the studies relied upon have concluded that gasoline has the same toxic effect as benzene, and none have concluded that the benzene component of gasoline is capable of *causing* AML. The studies relied upon by Plaintiffs make clear that the connection between gasoline or the benzene component of gasoline and AML is at this point in time only a hypothesis in need of further investigation. Based upon the court's review of these studies Plaintiffs' experts can only reliably attest to gasoline exposure as a theoretical or possible cause, not a probable cause of Henricksen's AML. *See Saldana v. Kmart Corp*., 260 F.3d 228, 234 (3d Cir. 2001) (“the mere possibility that something occurred in a particular way is not enough, as a matter of law, for a jury to find it probably happened that way”).

Though the court does not rely upon these cases in reaching this ruling, two other courts have reached the same general conclusion reached here. *Castellow v. Chevron USA*, 97 F.Supp.2d 780, 787 (S.D.Tex. 2000); *Parker v. Mobil Oil Corp*., 7 N.Y.3d 434, 824 N.Y.S.2d 584, 857 N.E.2d 1114 (2006). Plaintiffs did not discuss these cases. In *Castellow*, the family of a deceased gasoline service station manager brought suit against oil company, alleging that decedent's AML was caused by his occupational exposure to benzene and benzene-containing products, including gasoline**.** After reviewing Plaintiffs’ expert testimony and the scientific literature the court concluded “the opinions offered by Plaintiffs' expert witnesses are not reliable as they posit a theory that is not generally accepted (exposure to gasoline causes AML); that their particular hypothesis in this case has not been subjected to testing or peer review; and most importantly that, here, the result driven methodology (modeling to determine exposure assessment) is rife with error

and speculation." 97 F.Supp.2d at 797. In *Parker*, a service station attendant claimed that exposure to benzene on the job caused him to contract AML. The appellate court dismissed and reversed the lower court concluding summary judgment should have been granted because there was no evidence that benzene as a component of gasoline could proximately cause AML, and plaintiff's experts did not quantify the level of benzene to which plaintiff had been exposed on the job, or provide the dose-response relationship between benzene and AML.

Without reliable evidence of general causation, Plaintiffs' experts can not analyze specific causation while comporting with the scientific method. However, even if the court were to admit the testimony that gasoline is generally capable of causing AML, these studies do not support the conclusion that exposure at 8 ppm-years is capable of causing AML. Dr. Infante's specific causation opinion would also be subject to exclusion because the only quantitative measurement of exposure which he relied upon was Kaltofen's calculation, which the court has excluded as unreliable. Ct. Rec. 104, Ex. D at 308:4-7. The court's *Daubert* rulings leave Dr. Infante with no reliable dose/exposure data upon which to base his causation opinion, making his opinion also unreliable.

Though the law draws distinctions between general and specific causation, the court's ultimate focus is on the illness suffered by Henricksen and whether Plaintiffs can prove to a reasonable degree of certainty that his AML was caused by occupational exposure to gasoline. When questioned at his deposition whether it would be important to know Henricksen's dose for his opinions on AML, Infante replied: "Well, I mean, you know, I partially relied upon Kaltofen. I didn't like totally rely upon it. I mean, I -- in my -- you know, I could have done my report without Kaltofen's quantitative estimate of cumulative exposure." Ct. Rec. 104, Ex. D at 307:19-22. This statement gives the court some insight into Infante's methodology. If Infante could have prepared his report without any reliance upon any measurement of exposure, it appears Infante's methodology is more akin to Gardner's differential diagnosis concluding his occupational exposure was the likely cause of his AML based upon the lack of any other explanation

and Infante's belief that the occupation of tanker truck driver puts him an elevated risk of AML.

Although epidemiological evidence by itself can be sufficient to show causation, the absence of statistical support of causation is not necessarily fatal to a plaintiffs' case. *See Wells v. Ortho Pharmaceutical Corp*., 788 F.2d 741, 745 (11th Cir. 1986) (plaintiff's burden of proving that injuries were caused by the product did not necessarily require production of scientific studies showing a statistically significant association between the product and the injury in a large population); *Ferebee v. Chevron Chemical Co.*, 736 F.2d 1529 (D.C. Cir.), cert. denied, 469 U.S. 1062 (1984); Dore, A Commentary on the Use of Epidemiological Evidence in Demonstrating Cause-in-Fact, 7 HARV. ENVTL. L. REV. 429, 434 (1983) (courts may erroneously assume that a showing of no increased risk eliminates any possibility of causation but this may be related to difficulties inherent in epidemiology rather than the absence of a causal link). The court recognizes, as did the *Ferebee* court that "products liability law does not preclude recovery until a 'statistically significant' number of people have been injured or until science has had the time and resources to complete sophisticated laboratory studies of the chemical." *Ferebee*, 736 F.2d at 1535-36. Moreover, while the dose-response relationship is the primary way in toxic tort litigation of demonstrating causation, in some instances it may be possible to demonstrate exposure.

The lack of scientific support can not be overcome in this instance as this case does not offer indirect evidence of sufficient amount, specificity, and reliability to overcome the lack of direct evidence of causation. In this case, there is no actual exposure measurements and Plaintiffs have not countered Defendant's evidence that Henricksen's AML lacked the biological indicators typically characteristic of chemically induced AML. While in argument Henricksen's work environment has been described as "uniquely enclosed", there is no expert testimony on this aspect of Henricksen's work environment, nor is there a well-reasoned comparison of Henricksen's exposure to those existing in the scientific literature. The court is left with only his job description as a

tanker truck driver, which the court recognizes, the scientific literature demonstrates is a job category generally exposed to higher levels of benzene from gasoline than most job categories. This fact is insufficient to support the opinions offered. This analytical gap simply renders the expert testimony too speculative as a matter of law

G. SUMMARY JUDGMENT

General causation and specific causation are essential elements of Plaintiffs' prima facie case for each claim asserted in this litigation. *Norris*, 397 F.3d at 881. Expert testimony is necessary to make this showing since this is a toxic tort lawsuit. Without reliable expert testimony linking Henricksen's exposure to Defendant's gasoline to his subsequent development of AML, the jury could only speculate that a causal relationship exists. Such an inference would be impermissible. Therefore, Defendant's motions for summary judgment (Ct. Recs. 95, 100) must be and are **GRANTED**.

V. CONCLUSION

In conclusion, "[u]nder the regime of Daubert ... a district judge asked to admit scientific evidence must determine whether the evidence is genuinely scientific, as distinct from being unscientific speculation offered by a genuine scientist." *Rosen v. Ciba-Geigy Corp*., 78 F.3d 316, 318 (7th Cir. 1996). While the court does not question the scientific expertise of any of Plaintiffs' experts, their testimony must be excluded because their conclusions are not supported by the predominant scientific literature. While Plaintiffs' theory that the benzene in gasoline led to his development of AML has an air of plausibility about it, plausible hypotheses are not "scientific knowledge," *Daubert* on remand, 43 F.3d at 1315, but the building blocks and catalysts of such knowledge. Without evidence that "(at least) a recognized minority of scientists" in his field, *id*. at 1319, accept his methodology of inferring gasoline's ability to cause AML, Plaintiffs' causation opinions are based on subjective belief and unsupported speculation.

This ruling is not a comment on the qualifications of Plaintiffs' experts or their sincerity in their opinions or those of Mr. Henricksen as to what caused his AML. Herein the "law lags science; it does not lead it." *Rosen,* 78 F.3d at 319 (7th Cir. 1996).

Evidence that is an insightful hypothesis is not admissible in court if it lacks scientific rigor. Though Plaintiffs' theory may one day be validated through scientific research and experiment, the law today cannot apply that conjecture. As the Supreme Court explained,

> Law, [unlike science] must resolve disputes finally and quickly. The scientific project is advanced by broad and wide-ranging consideration of a multitude of hypotheses, for those that are incorrect will eventually be shown so, and that in itself is an advance. Conjectures that are probably wrong are of little use, however, in the project of reaching a quick, final, and binding legal judgment-often of great consequence-about a particular set of events in the past.... [I]n practice, a gatekeeping role for the judge, no matter how flexible, inevitably on occasion will prevent the jury from learning of authentic insights and innovations. That, nevertheless, is the balance that is struck by Rules of Evidence designed not for the exhaustive search for cosmic understanding but for the particularized resolution of legal disputes.

Daubert I, 509 U.S. at 597.

For the reasons stated above, its is hereby ordered:

1. Defendant's Motion to Exclude Opinions of Dr. Caton Based on Disclosure Violations (Ct. Rec. 67) is **GRANTED**.

2. Defendant's Motion in Limine to Limit the Testimony of Plaintiffs' Treating Physicians (Ct. Rec. 71) is **GRANTED**.

3. Defendant's Motion to Exclude the Testimony of Plaintiffs' Expert Witness Frank Gardner (Ct. Rec. 75) is **GRANTED**.

4. Defendant's Motion to Exclude Dose Opinions of Expert Sawyer due to Disclosure Violations (Ct. Rec. 79) is **DENIED**.

5. Defendant's Motion to Exclude Testimony of Plaintiffs' Expert Witness William Sawyer (Ct. Rec. 83) is **GRANTED**.

6. Defendant's Motion to Exclude the Testimony of Plaintiffs' Expert Witness Marco Kaltofen (Ct. Rec. 87) is **GRANTED**.

7. Defendant's Motion to Exclude Causation Opinions Based on Unreliable Epidemiological Evidence (Ct. Rec. 91) is **GRANTED**.

8. Defendant's Motion for Summary Judgment on General Causation (Ct. Rec. 95) is **GRANTED**.

9. Defendant's Motion for Summary Judgment on Specific Causation (Ct. Rec.

100) is **GRANTED**.

10. Defendant's Motion to Exclude Plaintiffs' Expert John Caton, M.D. (Ct. Rec. 127) is **DENIED as MOOT** in light of the above rulings; and

11. Plaintiff's expedited Motion to Supplement the Record (Ct. Rec. 160) is **GRANTED**.

The District Court Executive is directed to enter this order, enter judgment dismissing the First Amended Complaint and the claims therein with prejudice, and close this file.

IT IS SO ORDERED.

Dated this 11th day of February, 2009.

s/ Justin L. Quackenbush
JUSTIN L. QUACKENBUSH
SENIOR UNITED STATES DISTRICT JUDGE